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Prior Experiences of Racial Discrimination Impact Acute Resting-state Connectivity of the Bnst as a Predictor of PTSD in Black Adults

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PRIOR EXPERIENCES OF RACIAL DISCRIMINATION IMPACT ACUTE RESTING-STATE CONNECTIVITY OF THE BNST AS A PREDICTOR OF PTSD IN BLACK ADULTS

by

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ABSTRACT

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Altered resting-state activity of the bed nucleus of the stria terminalis (BNST) – which mediates anxious arousal and threat monitoring – is implicated in the etiology of posttraumatic stress disorder (PTSD). Experiences of racial discrimination can also increase one's risk for developing PTSD by eliciting chronic states of hypervigilance, which impair essential resting-state processes related to fear extinction. Considering the frequency in which Black Americans experience racial discrimination, the current study investigated acute BNST resting-state functional connectivity as a predictor of future PTSD symptoms, as well as the impact of racial discrimination on the BNST as a predictor of PTSD. Black adults (N = 95) who experienced a traumatic injury were recruited from the emergency department. Data was collected at two time points: *(1)* two-weeks post-trauma, where participants underwent a resting-state fMRI scan, and their baseline PTSD symptoms and history of racial discrimination were assessed; *(2)* six-months post-trauma, where their PTSD symptoms were reassessed. Results indicated that two-week BNST resting-state connectivity prospectively predicted PTSD symptoms six-months post-trauma. Additionally, prior experiences of racial discrimination moderated the relationship between acute BNST resting-state connectivity and future PTSD symptoms. Thus, in the acute aftermath of a traumatic event, resting-state connectivity of the BNST could be a useful biomarker of risk for PTSD in Black Americans, particularly for individuals who have experienced more racial discrimination throughout their lifetime.

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Introduction

Post-traumatic stress disorder (PTSD) is a pervasive mental illness that impacts 7-10% of Black individuals in their lifetime (Jones et al., 2022). PTSD is broadly characterized by intrusive and recurring memories of a traumatic event, alterations in arousal and reactivity, avoidance of trauma-related stimuli, and negative alterations in cognition and mood (American Psychiatric Association, 2013). Symptoms can lead to severe impairment in social, occupational, educational, and functional domains of life (Jellestad et al., 2021; Miao et al., 2018; North et al., 2019). Although Black adults have a higher risk for developing PTSD after experiencing a trauma (Roberts et al., 2010), research has historically focused on White populations (Sibrava et al., 2019). Psychosocial factors, such as previous trauma exposure and experiences with racial discrimination, increase risk for developing PTSD, but the heterogenous nature of the disorder complicates etiological investigation (Bird et al., 2021; Jones et al., 2022; Galatzer-Levy et al., 2013). Alone, psychosocial factors only account for a small portion of the variance in risk for developing PTSD (Ozer et al., 2003; Brewin et al., 2000; McLaughlin et al., 2014), and neuroimaging research can augment our understanding by delineating neural mechanisms related to the etiology of PTSD.

The Impact of Racial Discrimination

Among all minoritized ethnoracial groups, Black Americans have the highest risk of developing PTSD after experiencing a traumatic event (Roberts et al., 2011). This increased conditional risk may result from a kindling effect caused by chronic experiences of racial discrimination (Carter, 2007; Waller, 2003). Akin to other stressors, racial discrimination activates the stress response and elicits negative states of physiological arousal (Joseph et al., 2021; Berger & Sarnyai, 2014). Consequently, habitual exposure to racial discrimination can increase allostatic load, alter neurobiological functioning, and enhance the sensitivity of one's threat detection system (Miller et al., 2021; Berger & Sarnyai, 2014; Fani et al., 2021). This can lead to persistent states of threat monitoring that burden one's autonomic nervous system and

impairs biological functions required to cope with a stressor (Repetti et al., 2011; Miller et al., 2017). Thus, kindling increases one's vulnerability to PTSD following traumatic exposure (Post et al., 1997; Giotakos, 2020).

Consistent with this biological mechanism of risk, chronic states of hypervigilance and hyperarousal are common responses to racial discrimination (Kirkinis et al., 2018; Carter et al., 2013; Carter et al., 2020). However, considering the frequency in which Black Americans experience racial discrimination (Joseph et al., 2021), this may reflect an "adaptive trade-off" that prioritizes short-term safety over the long-term consequences of chronic hypervigilance (Repetti et al., 2011).

Increased hypervigilance in response to chronic racial discrimination may stem from altered activity of the BNST, as McEwen and Chattarji (2007) found repeated stressor exposure to impact BNST functioning. Rodents that experience chronic stress exhibit neurochemical changes in the BNST related to HPA axis regulation, as well as an increased frequency of anxiety-related behaviors. Although the BNST has yet to be studied in the context of racial discrimination, given its role in vigilance and stress response, the BNST could also be involved in vigilance related to race-based stressors (Carter, 2007; Berger & Sarnyai, 2014; Avery et al., 2015).

PTSD, the BNST, and the Stress Response

Neurobiological literature posits a core feature of PTSD is a hyperactive stress response (i.e. fight-or-flight) to trauma reminders. In the aftermath of trauma, frequent activation of the stress response can lead to the development, maintenance, and exacerbation of PTSD symptoms (Pitman et al., 2012; Miao et al., 2018; Pole, 1996; Brinkmann et al., 2017[a]; Simmons et al., 2008; Grupe & Nitschke, 2013). However, increased arousal in response to threatening cues is not inherently problematic. The fight-or-flight response is an evolutionarily adaptive process that involves activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic branch of the autonomic nervous system when one is faced with a threat, such as a

trauma-related stressor (Dunlop & Wong, 2019; Leistner & Menke, 2020; McEwen, 2017). Activation of these systems increase arousal, which is essential for coping with a threat and promoting safety. But this response becomes maladaptive when individuals over-interpret ambiguous cues (e.g. large crowds) as threatening and their stress response is chronically activated in the absence of danger (Charmandari et al., 2005; Maren, 2022; Jones & Moller, 2011; Brinkmann et al., 2017[a]). Preclinical models suggest the bed nucleus of the stria terminalis (BNST), which is densely connected with the HPA axis (Miles & Maren, 2019), plays a critical role in the dysfunctional threat detection and threat response systems that characterize PTSD (Fox & Shackman, 2016; Somerville et al., 2010; Davis et al., 1997; Walker et al., 2003).

The BNST is responsible for detecting environmental cues that signal uncertain threat, as well as initiating and prolonging states of anxiety (i.e. arousal) in response to ambiguous stimuli (Lebow & Chen, 2016; Hulsman et al., 2021). In Black adults who are chronically exposed to racial discrimination and have experienced a traumatic event, BNST functioning may play a prominent role in both adaptative and maladaptive threat monitoring processes. However, due to the diminutive size of the human BNST (Theiss et al., 2017), most knowledge on its functioning is based on rodent and non-human primate models. With recent advances in neuroimaging technology, researchers have begun investigating human BNST activation in relation to threat monitoring and arousal (Awasthi et al., 2020; Rabellino et al., 2017; Brinkmann et al., 2017[a]; Herrmann et al., 2016). Current findings from human studies corroborate results from preclinical models, but there is still a large gap in knowledge on the role of the BNST in the etiology of PTSD.

BNST Neurocircuitry and Composition

The BNST is a subcortical structure located in the ventromedial basal forebrain. Diffusion tensor imaging (DTI) reveals dense white matter connections with the limbic system (i.e., amygdala, hippocampus, parahippocampal gyrus), and functional magnetic resonance imaging (fMRI) studies report resting-state connectivity with the aforementioned structures along

with the cingulate cortex, precuneus, insula, and medial prefrontal cortex (mPFC; Avery et al., 2015; Weis et al., 2019). The BNST is also composed of both efferent and afferent projections to and from surrounding limbic structures that relay and integrate emotion-related information (Miles & Maren, 2019).

Notably, some limbic structures associated with the BNST (i.e. hippocampus, PCC, mPFC, precuneus) are also members of the default mode network (DMN; Raichle, 2019; Buckner et al., 2008). Structures within the DMN display greater functional connectivity with one another during self-referential tasks, such as emotion processing, when one is at rest (Satpute & Lindquist et al., 2019; Pan et al., 2018). Decreased intrinsic resting-state connectivity of the DMN is associated with greater PTSD severity and is theorized to reflect a diminished ability to remain in a relaxed state due to post-traumatic alterations in arousal and reactivity (Akiki et al., 2017[a]; Akiki et al., 2017[b]; Harnett et al., 2021; Lanius et al., 2010; Abdallah et al., 2017). However, identification of the underlying neural mechanisms is needed to substantiate this theory. Hyperactivation of the BNST in the absence of a threat is theorized to reflect a state of hypervigilance (Sommerville et al., 2010; Knight & Depue, 2019; Jenks et al., 2020; Pang et al., 2021), and considering its dense connections with the nodes of the DMN, the BNST could be implicated in the relationship between DMN resting-state connectivity and PTSD symptom severity (Lebow & Chen, 2016; Weis et al., 2019).

The BNST is also highly concentrated with stress-related neuropeptide receptors, such as corticotropin-releasing factor (CRF), which initiate and prolong states of arousal (Miles & Maren, 2019). Accordingly, the BNST has dense excitatory and inhibitory projections to and from the hypothalamus to regulate HPA axis activity (Dong & Swanson, 2005; Forray & Gysling, 2004; Goode & Maren, 2017). The BNST also exhibits strong connectivity with basal ganglia structures (i.e., nucleus accumbens, putamen, caudate, pallidum) and nodes of the brainstem, which further implicates it in autonomic arousal and threat-response behaviors (Hammack et al., 2010; Lebow & Chen, 2016; Crestani et al., 2013).

Overall, robust connections with the limbic system, HPA axis, and autonomic nervous system allow the BNST to modulate a wide range of emotion and stress related processes. Since dysfunction within these systems are often associated with PTSD (Orr & Roth, 2000; Awasthi et al., 2020; Robinson et al., 2019), altered BNST connectivity could be a key biomarker in the etiology of PTSD.

Fear, Anxiety, and the BNST

To conceptualize the diversified responsibilities of the BNST, Lebow and Chen (2016) developed the valence surveillance theory. They posit the BNST "surveys" the environment for threat-related stimuli and assesses the valence of incoming information, while also processing emotion-related information from the limbic system. The BNST integrates this information, and through its high density of stress receptors, either activates, prolongs, or diffuses the stress response. Hyperarousal due to overactivation of the stress response is a hallmark trait of PTSD, and it can produce the similar, yet distinct, emotions of fear and anxiety. Fear refers to a heightened state of arousal with rapid onset once a threat is encountered (e.g. you encounter a bear in the woods) and quickly dissipates once the threat is gone. Meanwhile, anxiety is more tonic. It leads to a lower level of arousal than fear and is elicited by a diffuse or uncertain threat (e.g. car crash victim sitting in a car), or one that is distant in time and/or proximity (e.g. car crash victim preparing to drive to work; Robinson et al., 2019; Grupe & Nitschke, 2013; Davis et al., 2009). Due to the ambiguity of potentially threatening stimuli, anxiety tends to be more chronically dysregulated than fear. Early rodent research posited a double dissociation between the neural mechanisms of fear and anxiety. The BNST was originally believed to only mediate anxious responses to threat (Davis et al., 1997; Walker et al., 2003), but recent human neuroimaging research challenges this theory. While the BNST likely has a more prominent role in anxiety (Hulsman et al., 2021; Brinkmann et al., 2017), findings suggest it is also implicated in fear-related processes (Shackman & Fox, 2016; Figel et al., 2019).

Maladaptive fear and anxiety-related processes are at the core of PTSD. Despite this, most of the trauma neuroimaging literature is based on task-based designs centered around phasic arousal (e.g. fear conditioning and extinction paradigms). Consequently, less is known about the role of anxiety in the etiology of PTSD. Further investigation of the BNST using resting-state fMRI, which is devoid of a clear threat but the unfamiliar and semi-disorienting (e.g., loud scanner noises, cannot see surrounding environment) conditions can create an ambiguously threatening environment, can fill that gap in knowledge by identifying anxiety-related structures that signal risk for PTSD.

The Role of the BNST in PTSD Symptomatology

The combination of the high volume of stress-related neuropeptide receptors in the BNST (Miles & Maren, 2019; Hammack et al., 2010) along with its white matter projections to the limbic system, autonomic nervous system (i.e. basal ganglia), and HPA axis (Avery et al., 2015; Foray & Gysling, 2004; Lebow & Chen, 2016; Crestani et al., 2013) heavily implicates the role of the BNST in anxiety. Accordingly, individuals with panic disorder exhibit sustained BNST activation during uncertain threat tasks compared to healthy controls (Brinkmann et al., 2017[b]), while individuals with social anxiety disorder show sustained, increased BNST activation during the anticipation of aversive vs. neutral stimuli (Figel et al., 2019). These paradigms represent states of hypervigilance, or prolonged arousal and threat monitoring, which is also a prominent feature of PTSD (Kimble et al., 2013; Naim et al., 2015). Brinkmann et al. (2017[a]) sought to identify the role of the BNST during states of anticipatory anxiety via blood oxygen level dependent (BOLD) signaling to assess activity within the brain while participants anticipated an aversive noise stimulus. Individuals with PTSD exhibited greater and more prolonged activation of the BNST during threat anticipation compared to healthy controls. While these findings are promising, they are limited in scope since anticipatory anxiety does not always occur in response to a known future threat.

Future threats that are uncertain, diffuse, or vary in time and/or proximity also elicit tonic states of arousal (Grupe et al., 2013; Somerville et al., 2010; Mobbs et al., 2010). In rodents, exposure to environmental light mimics a diffuse threat experienced by humans. Although light is not directly harmful, it increases a rodent's risk of being spotted by a predator, which in turn elicits hypervigilance and safety promoting behaviors (Hulsman et al., 2021). BNST functioning appears to mediate the behavior of light-exposed rodents, as Walker et al. (2003) found that rats with lesions of the BNST do not exhibit anxious behavior when exposed to environmental light, while those with an intact BNST do. This BNST-mediated response may also translate to humans.

To test the role of the human BNST in hypervigilance, Somerville et al. (2010) designed a future-oriented threat task where healthy participants viewed a pre-recorded video of a continuous line that fluctuated in height over time. Participants were informed when the height of the line reached a certain threshold, they would accumulate electrical shocks to be administered at a later time. Neural activity was measured using the BOLD signal, and skin conductance and heart rate were also measured to verify arousal during the task. Results reflected those from rodent models: BNST activation and autonomic arousal increased as the threat approached. These findings support the proposed dual involvement in threat monitoring (i.e., proximity of the threat) and threat response (i.e. increased arousal). Individuals with higher trait anxiety also experienced heightened BNST activation and autonomic arousal throughout the task, which indicates the BNST could serve as a useful neural marker of risk for anxiety-implicated disorders, such as PTSD.

Other studies have also connected BNST activity to either the presence of PTSD, or symptoms of PTSD. Awasthi et al. (2020) used an emotional word paradigm to assess BNST involvement in processing negative emotions related to trauma. They found heightened BNST activity in response to trauma vs. neutral words within the PTSD group but not healthy controls. Meanwhile, Rabellino et al. (2017) examined resting-state functional connectivity differences

between healthy controls and individuals with PTSD. They identified a significant relationship between BNST resting state activity and PTSD, as individuals with PTSD, compared to healthy controls, exhibited greater BNST activation. Still, no human study has used the BNST to prospectively predict the development of PTSD, leaving a fundamental gap in knowledge on the role of the BNST in the etiology of PTSD.

Current Study

This study was the first to use a prospective longitudinal design to investigate acute BNST resting-state functional connectivity as a predictor of PTSD symptoms six-months post-trauma within a sample of Black adults. Considering dysfunctional anxiety-related resting-state processes underlie the development of PTSD, and the BNST plays a critical role in the neural circuitry of anxiety, it was hypothesized that two-week resting-state connectivity of the BNST would predict symptoms of PTSD. Additionally, the BNST typically exhibits patterns of resting-state connectivity with the DMN and limbic system in non-PTSD populations. Since both of these networks are also implicated in the etiology of PTSD, it was hypothesized that heightened resting-state connectivity between the BNST and nodes of the DMN and limbic system would predict future PTSD. Finally, given the effects of chronic stress on BNST functioning and PTSD susceptibility, lifetime experiences with racial discrimination and prior exposure to traumatic events were expected to independently moderate the relationship between acute BNST connectivity and six-month PTSD symptoms.

Methods

Participants

Two-hundred and fifteen adults (Black/African American, n = 95) were recruited from the emergency department (ED) of Froedtert Hospital in Milwaukee, Wisconsin. Individuals were either screened directly in the ED or via telephone shortly after discharge. Individuals were eligible for study participation if they were seen in the ED due to a traumatic event (as defined by the *Diagnostic and Statistical Manual* - 5th edition; DSM-5), right-handed, spoke English,

between the ages of 18-65, and were able to schedule their first study visit within two weeks of their trauma. Individuals were excluded for contraindications of MRI scanning (e.g. metal objects in body, current or planned pregnancy in the next 6 months), if they suffered a head injury more severe than a mild traumatic brain injury (mTBI) as measured by the Glasgow Coma Scale (Sternbach, 2000), or if the injury resulted in a loss of consciousness. Additional exclusion criteria included injuries resulting from self-inflicted harm, individuals with severe vision or hearing impairments, a history of psychotic or manic symptoms, antipsychotic medication prescription, or if they were on police hold.

Eligible participants were provided written informed consent prior to the study and were financially compensated for each visit. Data was collected at two time points: two-weeks (T1) and six-months (T2) post-trauma. The study was approved by the Medical College of Wisconsin Institutional Review Board.

PTSD Symptom Assessment

The self-report PTSD Checklist for the DSM-5 (PCL-5) was used to assess PTSD symptoms at T1 (Bovin et al., 2016). The PCL-5 is a 20-item self-report questionnaire (current sample Cronbach α = .94) that assesses the frequency and severity of PTSD symptoms one has experienced within the past month, rated on a scale from 0 (not at all) to 4 (extremely). All questions refer to the same prespecified index trauma, and scores range from 0-80, with higher scores indicating greater symptom severity.

At T2, PTSD symptoms were assessed by a team member trained to administer the Clinician Administered PTSD scale for the DSM-5 (CAPS-5; Weathers et al., 2018). The CAPS-5 is a 20-item structured interview (current sample Cronbach α = .92) that measures the frequency and severity of PTSD symptoms one has experienced within the past month. All questions refer to the same prespecified index trauma, and the interviewer scores each item on the questionnaire from 0 (e.g., never/no effort to avoid) to 4 (e.g., daily or almost daily/extreme

efforts to avoid) based on the information provided by the participant. Total scores range from 0-80, with higher scores indicating greater symptom severity. Both the PCL-5 and CAPS-5 are well-established, empirically validated methods of PTSD assessment (Blevins et al., 2015, Pupo et al., 2011).

Racial Discrimination

The Perceived Ethnic Discrimination Questionnaire – Community Version (PEDQ-CV; Brandolo et al., 2006) was used to evaluate lifetime exposure to racial discrimination. The PEDQ-CV consists of 17 items that assesses participants' prior experiences with racial discrimination across various settings. Participants respond on a scale from 1 (never) to 5 (very often). The scores across all items are averaged to create a total score.

Lifetime Trauma Exposure

The Life Events Checklist for the *Diagnostic and Statistical Manual of Mental Disorders Fifth Edition* (LEC-5; Gray et al., 2004) was used to evaluate lifetime exposure to traumatic events. Participants rated their experience (i.e., 0 = does not apply, 1 = happened to them, 2 = witnessed the event, 3 = learned about the event) with 16 different traumatic events. The scoring method developed by Weis et al. (2022) was implemented. Total scores were weighted according to one's proximity (i.e., happened to them vs learned about the event) to the traumatic event.

Imaging Acquisition

Images were collected on a Discovery MR750 3.0 Tesla-weighted scanner, using a GE 32-channel head-coil. High resolution T1-weighted images were acquired with the following parameters: field of view (FOV), 240 mm; matrix, 256 × 224; slice thickness, 1 mm; 150 slices; repetition time (TR)/echo time (TE), 8.2/3.2 seconds, flip angle, 12° ; voxel size, 1 × 0.938 × 0.938 mm. At T1, participants underwent an eight-minute resting-state fMRI (rs-fMRI) scan where they were instructed to stare at a black cross on the screen; 240 volumes were acquired

using the following parameters: FOV, 22.4 mm; matrix, 64 × 64; slice thickness, 3.5 mm; 41 sagittal slices; TR/TE, 2000/25 milliseconds; flip angle, 77°; voxel size, 3.5 × 3.5 × 3.5 mm

fMRI Preprocessing

Structural and resting-state images were preprocessed using the default pipeline in the CONN Toolbox 20, with SPM 12 and MatLab 2019b (MathWorks, 2019). The first 3 TRs were discarded to account for initial instability of MR environment. All remaining images were motion-corrected using a 6-parameter linear transformation, normalized to Montreal Neurological Institute template (MNI 152), and then spatially smoothed using a 4-mm full-width-at-half-maximum kernel. BNST seed activity was extracted after smoothing. During the first-level analyses, head motion parameters (along with their first-order derivatives), white matter signal, and cerebrospinal fluid signal were regressed out. If more than 20% of the resting-state volumes were scrubbed or the scan quality was deemed poor after visual inspection, the participant was removed from analyses.

Data Analysis

A seed-to-voxel whole brain analysis, correlating the mean BOLD signal from the BNST with all other voxels in the brain, was conducted. The BNST seed, manually segmented for 3T fMRI images by the Blackford laboratory (Theiss et al., 2017), was imported into the CONN toolbox. Each participant was visually inspected to ensure proper placement of the BNST seed. A second-level regression analysis was conducted in CONN to test our hypothesis examining acute resting-state connectivity of the BNST as a predictor of PTSD symptoms six-months post-trauma. Gender, age, and education are associated with risk for PTSD (Brewin et al., 2000) and were controlled for, in addition to baseline PTSD symptoms (i.e., PCL-5 total scores). The threshold for statistical significance was set at two-tailed $p < .05$, with a height threshold of $p < .001$ uncorrected, and a cluster-size threshold of an adjusted $p < .05$ false discovery rate (FDR)-corrected.

The BNST-ROI patterns that significantly predicted PTSD were identified, and z-scores for the strength of resting-state connectivity between the structures was extracted for each participant and imported in SPSS. Moderation effects for the PEDQ and LEC on the relationship between BNST-ROI and CAPS-5 scores were analyzed using the Process macro for SPSS (Hayes, 2022).

Results

Demographics

The current study used a sample composed entirely of individuals who identified as Black or African-American (*n* = 95). Participants' mean age was 34.04 (*SD* = 10.48), 54.7% (*n* = 52) identified as female, 72.6% experienced a motor vehicle accident, and 89.5% either graduated from high school or obtained their GED (see Table 1).

Table 1 *Participant Demographics*

Age, Mean (SD)	34.04 (10.48)
Gender	
Female	52 (54.7%)
Male	43 (45.3%)
Mechanism of Injury	
Motor vehicle crash	69 (72.6)
Assault/altercation	13 (13.7)
Other	13 (13.7)
Education	
No high school or GED	10 (10.5)
High school or GED	35 (37.8)
Some college, no degree	26 (27.4)
Associate degree	11 (11.6)
Bachelor's degree	8 (8.4)
Master's degree	1 (1.1)

PTSD, Racial Discrimination, and Lifetime Trauma

Participant PTSD symptoms two-weeks post-trauma accounted for 19.8% of the variance in symptoms at the six-month follow-up, $F(1,93) = 22.02$, $p < .001$. Those who reported more severe symptoms two-weeks post-trauma were more likely to experience symptoms six-months post-trauma ($r = .466$, $p < .001$). Additionally, participants who endorsed PTSD symptoms two-weeks post-trauma tended to have experienced more racial discrimination ($r = .486$, $p < .001$) and had a greater lifetime exposure to traumatic events ($r = .464$, $p < .001$). Both experiences of racial discrimination ($F(1,89) = 5.42$, $p = .022$) and lifetime exposure to trauma ($F(1,93) = 11.05$, $p = .001$) were predictive of six-month PTSD symptoms, accounting for 5.7% and 10.6% of the variance respectively.

Table 2 *Psychological Measures*

Measure	Score, M (SD)
2-week PCL-5	26.16 ±18.03
6-month CAPS-5	13.04 ± 12.52
PEDQ-CV	1.98 ± 0.83
LEC (Weighted Total)	30.13 ± 16.83

BNST Resting-State Connectivity

The group level regression analysis predicting six-month PTSD symptoms identified increased resting-state connectivity between the BNST and four regions of interest (ROI): a cluster spanning the precuneus and PCC (Pre/PCC; peak voxel: -10, -48, +22; size = 90 voxels; *p-FDR* = .018), only the PCC (peak: -08, -36, +36; size = 75; *p-FDR* = .02), left angular gyrus (LAG; peak: -46, -64, +20; size = 70; *p-FDR* = .02), and hippocampus (peak: -18, -38, +02; size = 56; *p-FDR* = .03 7).

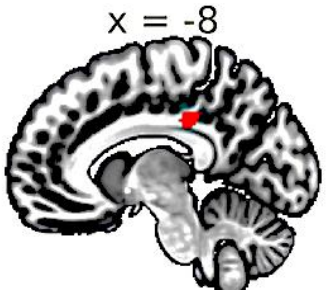
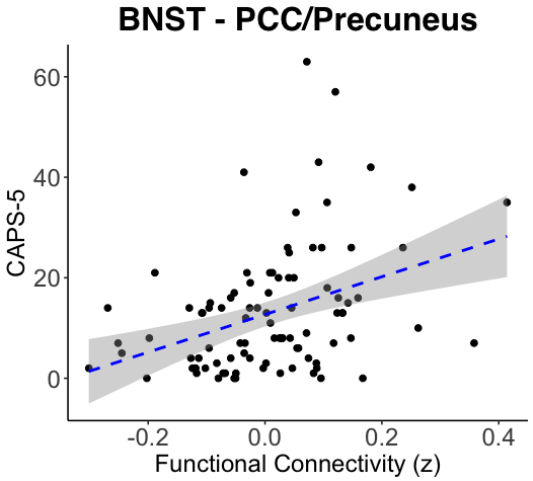


Figure 1. Sagittal view of the cluster spanning the PCC and Precuneus; corresponding graph of two-week BNST-Pre/PCC resting-state connectivity predicting 6-month CAPS-5 scores



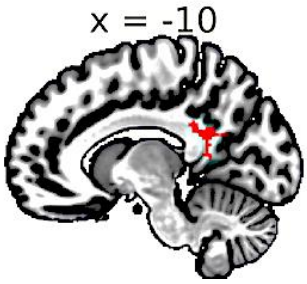
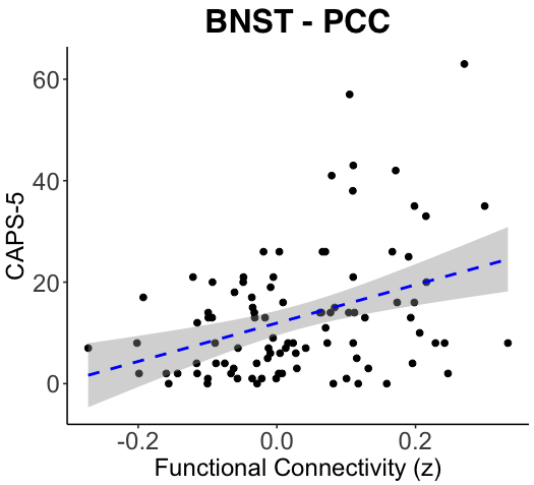


Figure 2. Sagittal view of cluster located on the PCC; corresponding graph of two-week BNST–PCC resting-state connectivity predicting 6-month CAPS-5 scores



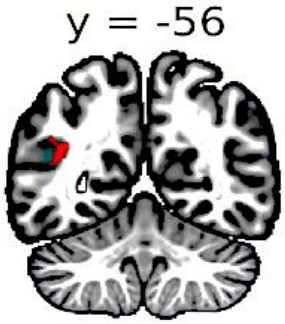
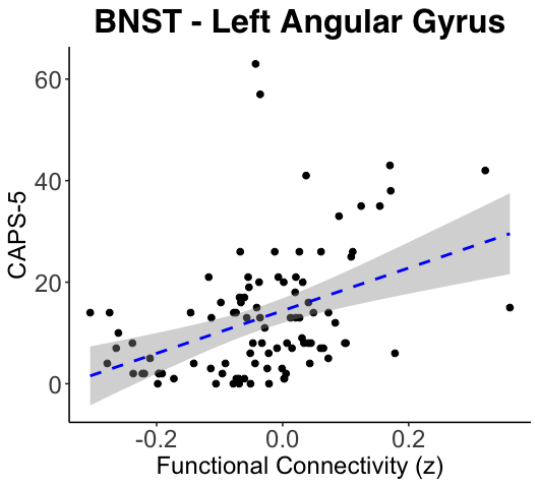
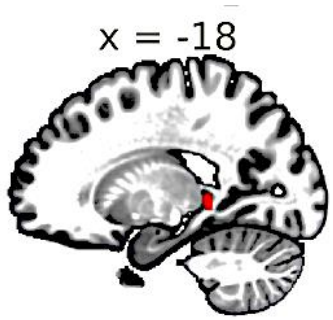


Figure 3. Coronal view of cluster located on the LAG; corresponding graph of two-week BNST–LAG resting-state connectivity predicting 6-month CAPS-5 scores





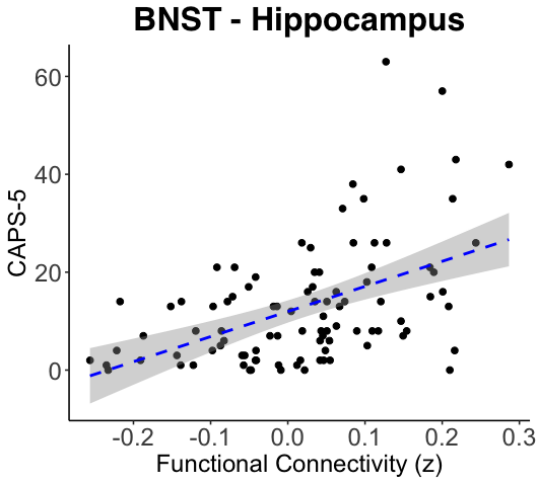


Figure 4. Sagittal view of cluster located on the hippocampus; correspond graph of two-week BNST–hippocampus resting-state connectivity predicting 6-month CAPS-5 scores

Racial discrimination was a significant moderator of the relationship between acute BNST resting-state connectivity and six-month PTSD symptoms. As experiences of racial discrimination increased, each BNST-ROI connection strengthened as a predictor of six-month PTSD symptoms (see tables 3-6): Pre/PCC (b = 26.46, $t(87)$ = 2.29, p = .025), PCC (b = 30.99, $t(87)$ = 2.55, p = .013), LAG (b = 25.33, $t(87)$ = 2.36, p = .021), hippocampus (b = 26.08, $t(87)$ = 2.81, p = .01). Lifetime exposure to traumatic events also moderated the relationship between acute BNST resting-state connectivity and six-month PTSD symptoms. As the frequency and severity (i.e. directly experiencing vs. learning of an event) of prior traumatic exposure increased, acute BNST-LAG (b = 1.38, $t(91)$ = 2.47, p = .01) and BNST-hippocampus (b = 1.16, $t(91)$ = 1.97, p = .05) resting-state connectivity strengthened as predictors of six-month PTSD symptoms (see tables 7 and 8).

Table 3 *PEDQ moderation of BNST-Pre/PCC predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
Pre/PCC	38.07	8.53	4.47	>.001
PEDQ	3.56	1.31	2.72	.01
Pre/PCC x PEDQ	26.36	11.53	2.29	.02
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	16.12	11.82	1.36	.18
0 SD	38.08	8.53	4.47	>.001
+1 SD	60.03	13.79	4.35	>.001

Table 4 *PEDQ moderation of BNST-PCC predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
PCC	36.16	9.07	3.99	>.001
PEDQ	3.40	1.31	2.60	.01
PCC x PEDQ	30.99	12.16	2.55	.01
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	10.35	12.05	.86	.39
0 SD	36.16	9.07	3.99	>.001
+1 SD	61.97	14.99	4.14	>.001

Table 5 *PEDQ moderation of BNST-LAG predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
LAG	42.97	8.35	5.15	>.001
PEDQ	3.46	1.25	2.77	.01
LAG x PEDQ	35.33	10.74	2.36	.02
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	21.88	11.85	1.85	.07
0 SD	42.97	8.35	5.15	>.001
+1 SD	64.07	12.60	5.08	>.001

Table 6 *PEDQ moderation of BNST-Hippocampus predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
Hippocampus	43.50	8.31	5.23	>.001
PEDQ	3.73	1.20	3.10	.003
Hippocampus x PEDQ	26.08	9.28	2.81	.006
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	21.78	12.11	1.80	.08
0 SD	43.50	8.31	5.24	>.001
+1 SD	65.22	10.53	6.20	>.001

Table 7 *LEC moderation of BNST-LAG predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
LAG	40.24	9.58	4.20	>.001
LEC	.12	.07	1.63	.11
LAG x LEC	1.38	.56	2.47	.02
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	17.01	12.27	1.39	.17
0 SD	40.24	9.58	4.20	>.001
+1 SD	63.48	14.48	4.38	>.001

Table 8 *LEC moderation of BNST-Hippocampus predicting CAPS-5 scores*

	b	*SE*	*t*	*p*
Outcome – CAPS-5 Total				
Hippocampus	47.98	9.04	5.31	>.001
LEC	.15	.07	2.29	.03
Hippocampus x LEC	1.16	.59	1.97	.05
	b	*SE*	*t*	*p*
Conditional Effects				
-1 SD	28.45	13.16	2.16	.03
0 SD	47.98	9.04	5.31	>.001
+1 SD	67.51	13.71	4.93	>.001

Discussion

Research on the role of the BNST in the etiology of PTSD is lacking despite its well-known implications in threat detection and anxious arousal (Lebow & Chen, 2016; Davis et al., 2009; Avery et al. 2015). Not only was this the first study to prospectively investigate the BNST as a predictor of future PTSD symptoms, but it was also the first time anyone has examined the relationship between the BNST, PTSD, and racial discrimination in a sample composed of only Black adults. Findings suggest the BNST can serve as a key biomarker of risk among recently traumatically injured Black Americans. Specifically, participants who exhibited increased BNST resting-state connectivity with nodes of the DMN (i.e., PCC, precuneus, left angular gyrus) and limbic system (i.e., hippocampus) acutely post-trauma were at a greater risk for developing PTSD at the six-month follow-up. This risk was independently moderated by lifetime history of racial discrimination and traumatic exposure, where acute BNST resting-state connectivity was a stronger predictor of future PTSD symptoms for individuals who experienced more racial discrimination or had a more severe history of traumatic exposure.

Among all ethnoracial groups in the United States, Black Americans are at the highest risk for developing PTSD, and prior experiences of racial discrimination increases their likelihood of developing severe symptoms (Roberts et al., 2010; Bird et al., 2021; Miller et al., 2021). Currently, the DSM-5 does not classify racial discrimination as a traumatic stressor, although some have argued for its inclusion (Holmes et al., 2016) because it is proven to have deleterious psychological effects that mirror PTSD, such as chronic hypervigilance (Carter, 2007; Carter et al., 2013; Berger & Sarnyai, 2014). Accordingly, racial discrimination is also connected to alterations in resting-state activity in regions associated with threat detection and arousal (Clark et al., 2021; Webb et al., 2022). Findings from the current study highlight a relationship between racial discrimination and BNST resting-state activity.

Heightened activity of the BNST in the absence of an immediate threat is associated with increased arousal and is theorized to reflect a state of hypervigilance (Sommerville et al.,

2010; Knight & Depue, 2019; Jenks et al., 2020; Pang et al., 2021). Typically, it is functionally adaptive for BNST activity to increase when anticipating an adverse situation, as it supports threat monitoring and threat response processes (Lebow & Chen, 2016; Davis et al., 2010). However, Brinkmann et al. (2017[a]) found individuals with PTSD exhibit hyperactivation of the BNST in concordance with heightened threat monitoring and more severe anticipatory anxiety when anticipating a future threat. High anticipatory anxiety increases the likelihood that one will utilize avoidant strategies to cope with the stressor, which impairs fear extinction and safety learning, and ultimately increases risk for PTSD (North et al., 2019; Sripada et al., 2013; Bryant et al., 2000). Brinkmann et al. (2017[a]) also found that individuals with PTSD in a hypervigilant state display increased connectivity between the BNST and structures involved in threat monitoring and arousal. Similarly, alterations in BNST resting-state connectivity exhibited by participants from the current study may also reflect a state of hypervigilance.

Hypervigilance also occurs in situations when the risk to encounter a threat is unknown. Although the current study did not measure participants' anxiety during the resting-state scan, altered BNST connectivity could reflect a state of hypervigilance induced by the conditions of the scanner. Resting-state fMRI is devoid of a clear threat, but the unfamiliar and semi-disorienting conditions (e.g., loud scanner noises, cannot see surrounding environment) can create an ambiguously threatening environment. Identifying safety cues can alleviate anxiety in these settings; however, individuals at risk for PTSD struggle to do so (Christianson et al., 2012; Jovanovic & Norrholm, 2011). Consistent failure to identify safe contextual features leads to chronic BNST activation and hypervigilance in the absence of danger, which disrupts critical resting-state processes that support fear extinction. Since this increases risk for developing other symptoms of PTSD (Choi et al., 2012; Schell, 2004; Miller et al., 2017), hypervigilance could mechanistically underlie the relationship between acute BNST resting-state connectivity and six-month PTSD symptoms from the current study.

In general, individuals with (or at risk for) PTSD may spend less time actually “at rest” due to heightened BNST-DMN connectivity. Since arousal is antithetical to the function of the DMN, high intrinsic DMN resting-state connectivity is thought to reflect general patterns of relaxation (Akiki et al., 2017[b]; Abdallah et al., 2017). However, a meta-analysis revealed that people with PTSD consistently exhibit decreased DMN intrinsic resting-state connectivity compared to healthy controls (Koch et al., 2016). Rabellino et al. (2017) suggested that within individuals who have PTSD, alterations in BNST connectivity can interfere with resting-state DMN functioning. They also identified a connection between BNST resting-state connectivity and hypervigilant symptoms of PTSD, which would also preclude relaxation. Results from the current study suggest this disruption exists within Black individuals who experience chronic racial discrimination, and that this experience also results in spending less time “at rest”. Indeed, psychophysiological research highlights a pattern of elevated resting heart rate and greater heart rate variability among people with PTSD and Black Americans with a history of racial discrimination, which reflects dysregulation of the stress response and chronic hyperarousal (Berger & Sarnyai, 2014; Schneider & Schwerdtfeger, 2020; Smith et al., 2020; Stam, 2007). Thus, shortly after experiencing a traumatic event, heightened resting-state connectivity between the BNST and DMN nodes may reflect a shift in focus from internally directed processes to externally oriented threat monitoring

Although the DMN is primarily active during wakeful rest, the individual structures that compose the network have other functions. Specifically, the PCC, precuneus, and hippocampus, are associated with hypervigilant threat appraisal and fear expression (Bremner et al., 1999; Rougemont-Bucking et al., 2010). When these structures are recruited for externally oriented tasks, DMN intrinsic connectivity decreases, and essential resting-state functions are inhibited. For example, PCC-hippocampus resting-state connectivity is critical for fear extinction, but decreased time at rest impairs this process and increases risk for PTSD (Miller et al., 2017; Akiki et al., 2017[a]). During states of broad vigilance (i.e., searching for an

ambiguous/non-specified environmental cue), the PCC supports visuospatial processing and plays a modulatory role in preparing for a threat (Leech & Sharp, 2014; Leech et al., 2011; Bremner et al., 1999). Thus, hypervigilant states elicited by an ambiguous threat would lead to low PCC-hippocampus connectivity and high BNST-PCC connectivity to facilitate threat monitoring. If hypervigilance was chronic, there would be a significant impairment in fear extinction.

While hypervigilance is a prominent feature of PTSD (Forsyth & Carter, 2012; Fani et al., 2021; Robinson et al., 2019), racism-related vigilance refers to the persistent anticipation of and preparation for racial discrimination, which can occur covertly in a variety of settings (Hicken et al., 2013). Considering the alarming rates at which Black Americans experience racial discrimination and threat it poses to their psychological and physical health, racism-related vigilance is often a necessary coping skill (Carter et al., 2017; Carter et al., 2017; Joseph et al., 2021; Jones et al., 2022; Berger & Sarnyai, 2014). However, while this vigilance is necessary to monitor potential harm, it also comes at a cost. Racism-related vigilance persistently activates the stress response, and when it is not paired with other coping strategies, it can be taxing on one's psychosocial and biological resources, which increases vulnerability to adverse outcomes following acute traumatic exposure (Joseph et al., 2021; Forsyth & Carter, 2012; Berger & Sarnyai, 2014; Webb et al., 2022). This cumulative effect of chronic stress is also referred to as kindling (Waller, 2003; Post et al., 1997; Giotakos, 2020).

Kindling increases the sensitivity of one's biological threat detection system, such as the hippocampus and other structures involved in fear neurocircuitry, which is theorized to contribute to the development of PTSD (Smid et al., 2022; Stam 2007). A hyperresponsive threat detection system can lead to chronic levels of stress and more severe stress responses. Over time, stress increases allostatic load and alters neurobiological functioning, such as in the BNST to modify functioning in support of increased threat monitoring (McEwen & Chattarji, 2007; McEwen et al., 2017). This would explain why acute BNST resting-state connectivity was

a stronger predictor of PTSD for individuals who experienced more racial discrimination or had a greater lifetime history of traumatic events.

Limitations

The current study has a couple noteworthy limitations. First, most participants experienced a motor vehicle accident, which tends to have less severe outcomes than interpersonal traumas (Thomas et al., 2021). Less than 10% of the sample had clinically significant levels of PTSD at the six-month follow-up. While it is encouraging to see natural resilience, our results might be more robust if the sample was composed of individuals who only admitted to the emergency department after experiencing an interpersonal trauma. Additionally, the PEDQ and LEC are retrospective measures, which makes them susceptible to bias in the respondents' memory (Lalande & Bonanno, 2011). Nevertheless, our results are consistent with the literature on racial discrimination predicting PTSD, and the reliability is substantiated by our use of the CAPS-5, which is the "gold standard" measurement for PTSD (Pupo et al., 2011). Lastly, BNST functioning was only assessed post-trauma. Considering the proposed kindling effect of chronic racism, as well as the impact allostatic load has on the BNST, it's unknown whether the alterations in BNST resting-state connectivity were a result of the acute stressor, prior experiences of racial discrimination, or both.

Conclusion

Findings from the current study implicate the BNST in the etiology of PTSD. Specifically in the immediate aftermath of a traumatic event, the BNST could be a key biomarker used to identify Black adults who are at risk for developing PTSD. Our results also provide further evidence that altered BNST resting-state connectivity likely disrupts DMN intrinsic connectivity within people who have PTSD. Heightened BNST-DMN resting-state connectivity could be a neural underpinning of hypervigilance, which ultimately increases risk for PTSD. It is also imperative to consider racial discrimination when examining risk for PTSD in Black adults.

BNST resting-state connectivity was a stronger predictor of future PTSD symptomatology for individuals who experienced more racial discrimination These findings are consistent with prior research, which underscores the importance to not only consider racial discrimination when investigating the neural underpinnings of risk for PTSD, but across all domains of risk. Furthermore, it is essential for future research to consider the dialectic of the adaptive vs pathological nature of post-trauma responses within the context of minoritized ethnoracial groups. For example, while it appears racism-related vigilance is a trauma response that mirrors hypervigilance in PTSD, the necessity of this response is driven by frequent exposure to race-based harm.

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